In the claims:

Please amend the claims as follows:

1-73. **Cancelled**

74. **(Previously presented)** A pharmaceutical composition for treating a disorder in which TNFα activity is detrimental comprising an isolated human antibody, or an antigen-binding portion thereof, that dissociates from human TNFα with a K_d of 1×10^{-8} M or less and a K_{off} rate constant of 1×10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-7} M or less, and at least one additional therapeutic agent.

75. **(Previously presented)** The pharmaceutical composition of claim 74, wherein the isolated human antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_{off} rate constant of 5×10^{-4} s^{-1} or less.

76. **(Previously presented)** The pharmaceutical composition of claim 74, wherein the isolated human antibody, or antigen-binding portion thereof, dissociates from human TNFα with a K_{off} rate constant of 1×10^{-4} s^{-1} or less.

77. **(Previously presented)** The pharmaceutical composition of claim 74, wherein the isolated human antibody, or antigen-binding portion thereof, neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-8} M or less.

78. **(Previously presented)** The pharmaceutical composition of claim 74, wherein the isolated human antibody, or antigen-binding portion thereof, neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-9} M or less.

79. **(Previously presented)** The pharmaceutical composition of claim 74, wherein the isolated human antibody, or antigen-binding portion thereof, neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-10} M or less.

80. **(Previously presented)** The pharmaceutical composition of claim 74, wherein the isolated human antibody, or antigen-binding portion thereof, is a recombinant antibody, or antigen-binding portion thereof.

81. **(Previously presented)** The pharmaceutical composition of claim 74, wherein the isolated human antibody, or antigen-binding portion thereof, inhibits human TNFα-induced expression of ELAM-1 on human umbilical vein endothelial cells.

82. **(Previously presented)** The pharmaceutical composition of claim 74, wherein the isolated human antibody, or antigen-binding portion thereof, is D2E7.

83. **(Currently amended)** The pharmaceutical composition of claim 74, wherein the additional therapeutic agent is selected from the group consisting of non-steroidal anti-inflammatory drugs, cytokine suppressive anti-inflammatory drugs, CDP-571/BAY-10-3356, cA2, 75 kdTNFR-IgG , 55 kdTNFR-IgG, IDEC-CE9.1/SB 210396, DAB 486-IL-2, DAB 389-IL-2, Anti-Tac, IL-4, IL-10, IL-4 agonists, IL-10 agonists, IL-1RA, TNF-bp/s-TNFR, S284, R973401, MK-966, Iloprost, methotrexate, thalidomide, ~~thalidomide-related drugs,~~ leflunomide, tranexamic acid, T614, prostaglandin E1, Tenidap, Naproxen, Meloxicam, Piroxicam, Diclofenac, Indomethacin, Sulfasalazine, Azathioprine, ICE inhibitors, zap-70 inhibitors, lck inhibitors, VEGF inhibitors, VEGF-R inhibitors, corticosteroids, TNF-convertase inhibitors, anti-IL-12 antibodies, interleukin-11, interleukin-13, interleukin-17 inhibitors, gold, penicillamine, chloroquine, hydroxychloroquine, chlorambucil, cyclophosphamide, cyclosporin, anti-thymocyte globulin, anti-CD4 antibodies, CD5-toxins, orally-administered ~~peptides,~~ collagen, lobenzarit disodium, Cytokine Regulating Agents HP228 and HP466, ICAM-1 antisense phosphorothioate oligodeoxynucleotides, soluble complement receptor 1, prednisone, orgotein, glycosaminoglycan polysulphate, minocycline, anti-IL2R antibodies, marine lipids, botanical lipids, auranofin, phenylbutazone, meclofenamic acid, flufenamic acid, ~~intravenous~~

~~immune globulin~~, zileuton, mycophenolic acid, tacrolimus, sirolimus, amiprilose, cladribine, azaribine, budenoside, epidermal growth factor, aminosalicylates, 6-mercaptopurine, metronidazole, lipoxygenase inhibitors, mesalamine, olsalazine, balsalazide, antioxidants, thromboxane inhibitors, IL-1 receptor antagonists, anti-IL-1β monoclonal antibodies, anti-IL-6 monoclonal antibodies, growth factors, elastase inhibitors, pyridinyl-imidazole compounds, glucuronide-conjugated prodrugs of prednisolone, dexamethasone or budesonide, dextran-conjugated prodrugs of prednisolone, dexamethasone or budesonide, soluble complement receptor 1, slow-release mesalazine, antagonists of Platelet Activating Factor (PAF), ciprofloxacin, lignocaine, prednisolone, methylprednisolone, cyclophosphamide, 4-aminopyridine, tizanidine, interferon-β1a, interferon-β1b, Copolymer 1, hyperbaric oxygen, intravenous immunoglobulin, clabribine, hypertonic saline solutions, antibiotics, continuous hemofiltration, carbapenems, antagonists of TNFα, antagonists of IL-1β, antagonists of IL-6 antagonists of IL-8, ~~antagonists of cytokines such as TNFα, IL-1β, IL-6 and/or IL-8~~, SK&F 107647, tetravalent guanylhydrazone CNI-1493, Tissue Factor Pathway Inhibitor, PHP, iron chelators and chelates, ~~including~~ diethylenetriamine pentaacetic acid-iron (III) complex, lisofylline, PGG-Glucan, apolipoprotein A-1 reconstituted with lipids, chiral hydroxamic acids, anti-endotoxin antibodies, E5531, $rBPI_{21}$, Synthetic Anti-Endotoxin Peptides, surfactant replacement therapy and anti-IL-8 antibodies.

84. **(Currently amended)** A method for inhibiting human TNFα activity in a human subject suffering from a disorder in which TNFα activity is detrimental, comprising administering to the human subject an antibody and at least one additional therapeutic agent such that human TNFα activity is inhibited, wherein the antibody is an isolated human antibody, or an antigen-binding portion thereof, that dissociates from human TNFα with a K_d of 1×10^{-8} M or less and a K_{off} rate constant of 1×10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-7} M or less, ~~and~~

85. **(Previously presented)** A method for inhibiting human TNFα activity in a human subject suffering from a disorder in which TNFα activity is detrimental, comprising

administering to the human subject an antibody and at least one additional therapeutic agent such that human TNFα activity is inhibited, wherein the antibody is an isolated human antibody, or antigen-binding portion thereof, with the following characteristics:

a) dissociates from human TNFα with a K_{off} rate constant of 1×10^{-3} s^{-1} or less, as determined by surface plasmon resonance,

b) has a light chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8 or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8 and/or 9;

c) has a heavy chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11 or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11 and/or 12.

86. **(Previously presented)** A method for inhibiting human TNFα activity in a human subject suffering from a disorder in which TNFα activity is detrimental, comprising administering to the human subject an antibody and at least one additional therapeutic agent such that human TNFα activity is inhibited, wherein the antibody is an isolated human antibody, or an antigen binding portion thereof, with a light chain variable region (LCVR) comprising the amino acid sequence of SEQ ID NO: 1 and a heavy chain variable region (HCVR) comprising the amino acid sequence of SEQ ID NO: 2.

87. **(Previously presented)** A method for inhibiting human TNFα activity in a human subject suffering from a disorder in which TNFα activity is detrimental, comprising administering to the human subject an antibody and at least one additional therapeutic agent such that human TNFα activity is inhibited, wherein the antibody is D2E7.

88. **(Currently amended)** The method of any one of claims 84, ~~85, 86, or 87~~, wherein the additional therapeutic agent is selected from the group consisting of ~~non-steroidal anti-inflammatory drugs~~, cytokine suppressive anti-inflammatory drugs, CDP-57111/BAY-10-3356, cA2, 75 kdTNFR-IgG , 55 kdTNFR-IgG, IDEC-CE9.1/SB 210396, DAB 486-IL-2, DAB 389-IL-2, Anti-Tac, IL-4, IL-10, IL-4 agonists, IL-10 agonists, IL-1RA, TNF-bp/s-TNFR, S284,

R973401, MK-966, Iloprost, ~~methotrexate~~, ~~thalidomide, thalidomide-related drugs, leflunomide, tranexamic acid, T-614, prostaglandin-E1, Tenidap, Naproxen, Meloxicam, Piroxicam, Diclofenac, Indomethacin, Sulfasalazine, Azathioprine, ICE inhibitors, zap-70 inhibitors, lck inhibitors, VEGF inhibitors, VEGF-R inhibitors, corticosteroids, TNF-convertase inhibitors, anti-IL-12 antibodies~~, ~~interleukin-11, interleukin-13, interleukin-17 inhibitors, gold, penicillamine, chloroquine, hydroxychloroquine, chlorambucil, cyclophosphamide, cyclosporin, anti-thymocyte-globulin, anti-CD4 antibodies, CD5-toxins, orally-administered peptides, collagen, lobenzarit-disodium, Cytokine-Regulating-Agents HP228 and HP466, ICAM-1 antisense phosphorothioate oligodeoxynucleotides, soluble complement receptor-1, prednisone, orgotein, glycosaminoglycan-polysulphate, minocycline, anti-IL2R-antibodies, marine-lipids, botanical-lipids, auranofin, phenylbutazone, meclofenamic-acid, flufenamic-acid, intravenous immune-globulin, zileuton, mycophenolic-acid, tacrolimus, sirolimus, amiprilose, cladribine, azaribine, budenoside, epidermal-growth-factor, aminosalicylates, 6-mercaptopurine, metronidazole, lipoxygenase-inhibitors, mesalamine, olsalazine, balsalazide, antioxidants, thromboxane-inhibitors, IL-1-receptor-antagonists, anti-IL-1β-monoclonal-antibodies, anti-IL-6 monoclonal-antibodies, growth-factors, elastase-inhibitors, pyridinyl-imidazole-compounds, glucuronide-conjugated-prodrugs-of-prednisolone, dexamethasone-or-budesonide, dextran-conjugated-prodrugs-of-prednisolone, dexamethasone-or-budesonide, soluble-complement receptor-1, slow-release-mesalazine, antagonists-of-Platelet-Activating-Factor-(PAF), ciprofloxacin, lignocaine, prednisolone, methylprednisolone, cyclophosphamide, 4-aminopyridine, tizanidine, interferon-β1a, interferon-β1b, Copolymer-1, hyperbaric-oxygen, intravenous-immunoglobulin, clabribine, hypertonic-saline-solutions, antibiotics, continuous hemofiltration, carbapenems, antagonists-of-cytokines-such-as-TNFα, IL-1β, IL-6-and/or-IL-8, SK&F-107647, tetravalent-guanylhydrazone-CNI-1493, Tissue-Factor-Pathway-Inhibitor, PHP, iron-chelators-and-chelates, including-diethylenetriamine-pentaacetic-acid-iron-(III)-complex, lisofylline, PGG-Glucan, apolipoprotein-A-1-reconstituted-with-lipids, chiral-hydroxamic-acids, anti-endotoxin-antibodies, E5531, rBPI$_{21}$, Synthetic-Anti-Endotoxin-Peptides, surfactant replacement-therapy-and-anti-IL-8-antibodies~~.

89. **(Previously presented)** The method of any one of claims 84, 85, 86, or 87, wherein the disorder is rheumatoid arthritis.

90. **(Currently amended)** The method of claim 89, wherein the wherein the additional therapeutic agent is selected from the group consisting of ~~non-steroidal anti-inflammatory drugs~~, cytokine suppressive anti-inflammatory drugs, CDP-571/BAY-10-3356, cA2, 75 kdTNFR-IgG , 55 kdTNFR-IgG, IDEC-CE9.1/SB 210396, DAB 486-IL-2, DAB 389-IL-2, Anti-Tac, IL-4, IL-10, IL-4 agonists, IL-10 agonists, IL-1RA, TNF-bp/s-TNFR, S284, R973401, MK-966, Iloprost, ~~methotrexate,~~ thalidomide, ~~thalidomide-related drugs~~, ~~leflunomide,~~ tranexamic acid, T614, prostaglandin E1, Tenidap, Naproxen, Meloxicam, Piroxicam, ~~Diclofenac~~, Indomethacin, ~~Sulfasalazine,~~ Azathioprine, ICE inhibitors, zap-70 inhibitors, lck inhibitors, VEGF inhibitors, VEGF-R inhibitors, corticosteroids, TNF-convertase inhibitors, anti-IL-12 antibodies, interleukin-11, interleukin-13, interleukin-17 inhibitors, ~~gold,~~ penicillamine, ~~chloroquine, hydroxychloroquine~~, chlorambucil, cyclophosphamide, cyclosporin, anti-thymocyte globulin, anti-CD4 antibodies, CD5-toxins, orally-administered ~~peptides,~~ collagen, lobenzarit disodium, Cytokine Regulating Agents HP228 and HP466, ICAM-1 antisense phosphorothioate oligodeoxynucleotides, soluble complement receptor 1, ~~prednisone,~~ orgotein, glycosaminoglycan polysulphate, minocycline, anti-IL2R antibodies, marine lipids, botanical lipids, auranofin, phenylbutazone, meclofenamic acid, flufenamic acid, intravenous immunoglobulin ~~immune globulin~~, zileuton, mycophenolic acid, tacrolimus, sirolimus, amiprilose, cladribine, and azaribine.

91. **(Currently amended)** The method of any one of claims 84, ~~85, 86, or 87~~, wherein the disorder is inflammatory bowel disease.

92. **(Previously presented)** The method of claim 91, wherein the additional therapeutic agent is selected from the group consisting of budenoside, epidermal growth factor, aminosalicylates, 6-mercaptopurine, metronidazole, lipoxygenase inhibitors, mesalamine, olsalazine, balsalazide, antioxidants, thromboxane inhibitors, IL-1 receptor antagonists, anti-IL-1β monoclonal antibodies, anti-IL-6 monoclonal antibodies, growth factors, elastase inhibitors, pyridinyl-imidazole compounds, glucuronide-conjugated prodrugs of prednisolone, dexamethasone or budesonide, dextran-conjugated prodrugs of prednisolone, dexamethasone or

budesonide, soluble complement receptor 1, slow-release mesalazine, antagonists of Platelet Activating Factor (PAF), ciprofloxacin, and lignocaine.

93. **(Currently amended)** The method of any one of claims 84, ~~85, 86, or 87~~, wherein the disorder is multiple sclerosis.

94. **(Previously presented)** The method of claim 93, wherein the additional therapeutic agent is selected from the group consisting of corticosteroids, prednisolone, methylprednisolone, azathioprine, cyclophosphamide, cyclosporine, methotrexate, 4-aminopyridine, tizanidine, interferon-β1a, interferon-β1b, Copolymer 1, hyperbaric oxygen, intravenous immunoglobulin, clabribine, CDP-571/BAY-10-3356, cA2, 75 kdTNFR-IgG, 55 kdTNFR-IgG, IL-10, IL-4, and IL-10 agonists, and IL-4 agonists.

95. **(Currently amended)** The method of any one of claims 84, ~~85, 86, or 87~~, wherein the disorder is sepsis.

96. **(Previously presented)** The method of claim 95, wherein the additional therapeutic agent is selected from the group consisting of hypertonic saline solutions, antibiotics, intravenous gamma globulin, continuous hemofiltration, carbapenems, antagonists of TNFα, antagonists of IL-1β, antagonists of IL-6, antagonists of IL-8, CDP-571//BAY-10-3356, cA2, 75 kdTNFR-IgG, 55 kdTNFR-IgG, Cytokine Regulating Agents (CRAs) HP228 and HP466, SK&F 107647, tetravalent guanylhydrazone CNI-1493, Tissue Factor Pathway Inhibitor, PHP, iron chelators and chelates, ~~including~~ diethylenetriamine pentaacetic acid-iron (III) complex, lisofylline, PGG-Glucan, apolipoprotein A-1 reconstituted with lipids, chiral hydroxamic acids, anti-endotoxin antibodies, E5531, $rBPI_{21}$, and Synthetic Anti-Endotoxin Peptides.

97. **(Currently amended)** The method of any one of claims 84, ~~85, 86, or 87~~, wherein the disorder is adult respiratory distress syndrome (ARDS).

98. **(Previously presented)** The method of claim 97, wherein the additional therapeutic agent is selected from the group consisting of anti-IL-8 antibodies, surfactant replacement therapy, CDP-571/BAY-10-3356, cA2, 75 kdTNFR-IgG, and 55 kdTNFR-IgG.

99. **(Previously presented)** A method for treating a subject suffering from a disorder in which TNFα activity is detrimental, comprising administering to the subject an antibody and at least one additional therapeutic agent, such that the disorder is treated, wherein the antibody is an isolated human antibody, or an antigen-binding portion thereof, that dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less.

100. **(Previously presented)** A method for treating a subject suffering from a disorder in which TNFα activity is detrimental, comprising administering to the subject an antibody and at least one additional therapeutic agent such that the disorder is treated, wherein the antibody is an isolated human antibody, or antigen-binding portion thereof, with the following characteristics:

a) dissociates from human TNFα with a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, as determined by surface plasmon resonance;

b) has a light chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8 or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8 and/or 9;

c) has a heavy chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11 or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11 and/or 12.

101. **(Previously presented)** A method for treating a subject suffering from a disorder in which TNFα activity is detrimental, comprising administering to the subject an antibody and at least one additional therapeutic agent, such that the disorder is treated, wherein the antibody is an isolated human antibody, or an antigen binding portion thereof, with a light chain variable region (LCVR) comprising the amino acid sequence of SEQ ID NO: 1 and a heavy chain variable region (HCVR) comprising the amino acid sequence of SEQ ID NO: 2

102. **(Previously presented)** A method for treating a subject suffering from a disorder in which TNFα activity is detrimental, comprising administering to the subject an antibody and at least one additional therapeutic agent such that the disorder is treated, wherein the antibody is D2E7.

103. **(Currently amended)** The method of any one of claims 99, ~~100, 101, or 102~~, wherein the additional therapeutic agent is selected from the group consisting of wherein the additional therapeutic agent is selected from the group consisting of ~~non-steroidal anti-inflammatory drugs~~, cytokine suppressive anti-inflammatory drugs, CDP-57111/BAY-10-3356, cA2, 75 kdTNFR-IgG , 55 kdTNFR-IgG, IDEC-CE9.1/SB 210396, DAB 486-IL-2, DAB 389-IL-2, Anti-Tac, IL-4, IL-10, IL-4 agonists, IL-10 agonists, IL-1RA, TNF-bp/s-TNFR, S284, R973401, MK-966, Iloprost, ~~methotrexate~~, ~~thalidomide, thalidomide-related drugs, leflunomide, tranexamic acid, T-614, prostaglandin E1, Tenidap, Naproxen, Meloxicam, Piroxicam, Diclofenac, Indomethacin, Sulfasalazine, Azathioprine, ICE inhibitors, zap-70 inhibitors, lck inhibitors, VEGF inhibitors, VEGF-R inhibitors, corticosteroids, TNF-convertase inhibitors, anti-IL-12 antibodies~~, ~~interleukin-11, interleukin-13, interleukin-17 inhibitors, gold, penicillamine, chloroquine, hydroxychloroquine, chlorambucil, cyclophosphamide, cyclosporin, anti-thymocyte globulin, anti-CD4 antibodies, CD5-toxins, orally-administered peptides, collagen, lobenzarit disodium, Cytokine Regulating Agents HP228 and HP466, ICAM-1 antisense phosphorothioate oligodeoxynucleotides, soluble complement receptor 1, prednisone, orgotein, glycosaminoglycan polysulphate, minocycline, anti-IL2R antibodies, marine lipids, botanical lipids, auranofin, phenylbutazone, meclofenamic acid, flufenamic acid, intravenous immune globulin, zileuton, mycophenolic acid, tacrolimus, sirolimus, amiprilose, cladribine, azaribine, budenoside, epidermal growth factor, aminosalicylates, 6-mercaptopurine, metronidazole, lipoxygenase inhibitors, mesalamine, olsalazine, balsalazide, antioxidants, thromboxane inhibitors, IL-1 receptor antagonists, anti-IL-1β monoclonal antibodies, anti-IL-6 monoclonal antibodies, growth factors, elastase inhibitors, pyridinyl-imidazole compounds, glucuronide-conjugated prodrugs of prednisolone, dexamethasone or budesonide, dextran-conjugated prodrugs of prednisolone, dexamethasone or budesonide, soluble complement receptor 1, slow-release mesalazine, antagonists of Platelet Activating Factor (PAF), ciprofloxacin, lignocaine, prednisolone, methylprednisolone, cyclophosphamide, 4-~~

~~aminopyridine, tizanidine, interferon-β1a, interferon-β1b, Copolymer 1, hyperbaric oxygen, intravenous immunoglobulin, clabribine, hypertonic saline solutions, antibiotics, continuous hemofiltration, carbapenems, antagonists of cytokines such as TNFα, IL-1β, IL-6 and/or IL-8, SK&F 107647, tetravalent guanylhydrazone CNI-1493, Tissue Factor Pathway Inhibitor, PHP, iron chelators and chelates, including diethylenetriamine pentaacetic acid iron (III) complex, lisofylline, PGG-Glucan, apolipoprotein A-1 reconstituted with lipids, chiral hydroxamic acids, anti-endotoxin antibodies, E5531, $rBPI_{21}$, Synthetic Anti-Endotoxin Peptides, surfactant replacement therapy and anti-IL-8 antibodies~~.

104. **(Previously presented)** The method of any one of claims 99, 100, 101, or 102, wherein the disorder is rheumatoid arthritis.

105. **(Currently amended)** The method of claim 104, wherein the wherein the additional therapeutic agent is selected from the group consisting of ~~non-steroidal anti-inflammatory drugs~~, cytokine suppressive anti-inflammatory drugs, CDP-571/BAY-10-3356, cA2, 75 kdTNFR-IgG , 55 kdTNFR-IgG, IDEC-CE9.1/SB 210396, DAB 486-IL-2, DAB 389-IL-2, Anti-Tac, IL-4, IL-10, IL-4 agonists, IL-10 agonists, IL-1RA, TNF-bp/s-TNFR, S284, R973401, MK-966, Iloprost, ~~methotrexate,~~ thalidomide, ~~thalidomide-related drugs~~, ~~leflunomide,~~ tranexamic acid, T614, prostaglandin E1, Tenidap, Naproxen, Meloxicam, Piroxicam, ~~Diclofenac~~, Indomethacin, ~~Sulfasalazine,~~ Azathioprine, ICE inhibitors, zap-70 inhibitors, lck inhibitors, VEGF inhibitors, VEGF-R inhibitors, corticosteroids, TNF-convertase inhibitors, anti-IL-12 antibodies, interleukin-11, interleukin-13, interleukin-17 inhibitors, ~~gold,~~ penicillamine, ~~chloroquine, hydroxychloroquine~~, chlorambucil, cyclophosphamide, cyclosporin, anti-thymocyte globulin, anti-CD4 antibodies, CD5-toxins, orally-administered ~~peptides,~~ collagen, lobenzarit disodium, Cytokine Regulating Agents HP228 and HP466, ICAM-1 antisense phosphorothioate oligodeoxynucleotides, soluble complement receptor 1, ~~prednisone,~~ orgotein, glycosaminoglycan polysulphate, minocycline, anti-IL2R antibodies, marine lipids, botanical lipids, auranofin, phenylbutazone, meclofenamic acid, flufenamic acid, intravenous immunoglobulin ~~immune globulin~~, zileuton, mycophenolic acid, tacrolimus, sirolimus, amiprilose, cladribine, and azaribine.

106. **(Currently amended)** The method of any one of claims 99, ~~100, 101, or 102~~, wherein the disorder is inflammatory bowel disease.

107. **(Previously presented)** The method of claim 106, wherein the additional therapeutic agent is selected from the group consisting of budenoside, epidermal growth factor, aminosalicylates, 6-mercaptopurine, metronidazole, lipoxygenase inhibitors, mesalamine, olsalazine, balsalazide, antioxidants, thromboxane inhibitors, IL-1 receptor antagonists, anti-IL-1β monoclonal antibodies, anti-IL-6 monoclonal antibodies, growth factors, elastase inhibitors, pyridinyl-imidazole compounds, glucuronide-conjugated prodrugs of prednisolone, dexamethasone or budesonide, dextran-conjugated prodrugs of prednisolone, dexamethasone or budesonide, soluble complement receptor 1, slow-release mesalazine, antagonists of Platelet Activating Factor (PAF), ciprofloxacin, and lignocaine.

108. **(Currently amended)** The method of any one of claims 99, ~~100, 101, or 102~~, wherein the disorder is multiple sclerosis.

109. **(Previously presented)** The method of claim 108, wherein the additional therapeutic agent is selected from the group consisting of corticosteroids, prednisolone, methylprednisolone, azathioprine, cyclophosphamide, cyclosporine, methotrexate, 4-aminopyridine, tizanidine, interferon-β1a, interferon-β1b, Copolymer 1, hyperbaric oxygen, intravenous immunoglobulin, clabribine, CDP-571/BAY-10-3356, cA2, 75 kdTNFR-IgG, 55 kdTNFR-IgG, IL-10, IL-4, and IL-10 agonists, and IL-4 agonists.

110. **(Currently amended)** The method of any one of claims 99, ~~100, 101, or 102~~, wherein the disorder is sepsis.

111. **(Currently amended)** The method of claim 110, wherein the additional therapeutic agent is selected from the group consisting of hypertonic saline solutions, antibiotics, intravenous gamma globulin, continuous hemofiltration, carbapenems, antagonists of TNFα, antagonists of IL-1β, antagonists of IL-6, antagonists of IL-8, CDP-571//BAY-10-3356, cA2, 75 kdTNFR-IgG, 55 kdTNFR-IgG, Cytokine Regulating Agents (CRAs) HP228 and HP466, SK&F 107647, tetravalent guanylhydrazone CNI-1493, Tissue Factor Pathway Inhibitor, PHP, iron

chelators and chelates, ~~including~~ diethylenetriamine pentaacetic acid-iron (III) complex, lisofylline, PGG-Glucan, apolipoprotein A-1 reconstituted with lipids, chiral hydroxamic acids, anti-endotoxin antibodies, E5531, $rBPI_{21}$, and Synthetic Anti-Endotoxin Peptides.

112. **(Currently amended)** The method of any one of claims 99, ~~100, 101, or 102~~, wherein the disorder is adult respiratory distress syndrome (ARDS).

113. **(Previously presented)** The method of claim 112, wherein the additional therapeutic agent is selected from the group consisting of anti-IL-8 antibodies, surfactant replacement therapy, CDP-571/BAY-10-3356, cA2, 75 kdTNFR-IgG, and 55 kdTNFR-IgG.

114. **(Previously presented)** A method for inhibiting human TNFα activity in a human subject suffering from a disorder in which TNFα activity is detrimental, wherein the disorder is selected from the group consisting of periodontal disease, obesity, and radiation toxicity, comprising administering to the human subject an antibody such that human TNFα activity in the human subject is inhibited, wherein the antibody is an isolated human antibody, or an antigen-binding portion thereof, that dissociates from human TNFα with a K_d of 1×10^{-8} M or less and a K_{off} rate constant of 1×10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-7} M or less.

115. **(Previously presented)** A method for inhibiting human TNFα activity in a human subject suffering from a disorder in which TNFα activity is detrimental, wherein the disorder is selected from the group consisting of periodontal disease, obesity, and radiation toxicity, comprising administering to the human subject an antibody such that human TNFα activity in the human subject is inhibited, wherein the antibody is an isolated human antibody, or antigen-binding portion thereof, with the following characteristics:

a) dissociates from human TNFα with a K_{off} rate constant of 1×10^{-3} s^{-1} or less, as determined by surface plasmon resonance;

b) has a light chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8 or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8 and/or 9;

c) has a heavy chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11 or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11 and/or 12.

116. **(Previously presented)** A method for inhibiting human TNFα activity in a human subject suffering from a disorder in which TNFα activity is detrimental, wherein the disorder is selected from the group consisting of periodontal disease, obesity, and radiation toxicity, comprising administering to the human subject an antibody such that human TNFα activity in the human subject is inhibited, wherein the antibody is an isolated human antibody, or an antigen binding portion thereof, with a light chain variable region (LCVR) comprising the amino acid sequence of SEQ ID NO: 1 and a heavy chain variable region (HCVR) comprising the amino acid sequence of SEQ ID NO: 2.

117. **(Previously presented)** A method for inhibiting human TNFα activity in a human subject suffering from a disorder in which TNFα activity is detrimental, wherein the disorder is selected from the group consisting of periodontal disease, obesity, and radiation toxicity, comprising administering to the human subject an antibody such that human TNFα activity in the human subject is inhibited, wherein the antibody is D2E7.

118. **(Previously presented)** A method for treating a subject suffering from a disorder in which TNFα activity is detrimental, wherein the disorder is selected from the group consisting of periodontal disease, obesity, and radiation toxicity, comprising administering to the subject an antibody such that the disorder is treated, wherein the antibody is an isolated human antibody, or an antigen-binding portion thereof, that dissociates from human TNFα with a k_d of 1×10^{-8} m or less and a k_{off} rate constant of 1×10^{-3} s^{-1} or less, both determined by surface

plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-7} m or less.

119. **(Previously presented)** A method for treating a subject suffering from a disorder in which TNFα activity is detrimental, wherein the disorder is selected from the group consisting of periodontal disease, obesity, and radiation toxicity, comprising administering to the subject an antibody such that the disorder is treated, wherein the antibody is an isolated human antibody, or antigen-binding portion thereof, with the following characteristics:

a) dissociates from human TNFα with a K_{off} rate constant of 1×10^{-3} s^{-1} or less, as determined by surface plasmon resonance;

b) has a light chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8 or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8 and/or 9;

c) has a heavy chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11 or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11 and/or 12.

120. **(Previously presented)** A method for treating a subject suffering from a disorder in which TNFα activity is detrimental, wherein the disorder is selected from the group consisting of periodontal disease, obesity, and radiation toxicity, comprising administering to the subject an antibody such that the disorder is treated, wherein the antibody is an isolated human antibody, or an antigen binding portion thereof, with a light chain variable region (LCVR) comprising the amino acid sequence of SEQ ID NO: 1 and a heavy chain variable region (HCVR) comprising the amino acid sequence of SEQ ID NO: 2

121. **(Previously presented)** A method for treating a subject suffering from a disorder in which TNFα activity is detrimental, wherein the disorder is selected from the group consisting of periodontal disease, obesity, and radiation toxicity, comprising administering to the subject an antibody such that the disorder is treated, wherein the antibody is D2E7.

122. **(New)** The method of claim 118, wherein the additional therapeutic agent is selected from the group consisting of non-steroidal anti-inflammatory drugs, cytokine suppressive anti-inflammatory drugs, CDP-571/BAY-10-3356, cA2, 75 kdTNFR-IgG , 55 kdTNFR-IgG, IDEC-CE9.1/SB 210396, DAB 486-IL-2, DAB 389-IL-2, Anti-Tac, IL-4, IL-10, IL-4 agonists, IL-10 agonists, IL-1RA, TNF-bp/s-TNFR, S284, R973401, MK-966, Iloprost, methotrexate, thalidomide, leflunomide, tranexamic acid, T614, prostaglandin E1, Tenidap, Naproxen, Meloxicam, Piroxicam, Diclofenac, Indomethacin, Sulfasalazine, Azathioprine, ICE inhibitors, zap-70 inhibitors, lck inhibitors, VEGF inhibitors, VEGF-R inhibitors, corticosteroids, TNF-convertase inhibitors, anti-IL-12 antibodies, interleukin-11, interleukin-13, interleukin-17 inhibitors, gold, penicillamine, chloroquine, hydroxychloroquine, chlorambucil, cyclophosphamide, cyclosporin, anti-thymocyte globulin, anti-CD4 antibodies, CD5-toxins, orally-administered collagen, lobenzarit disodium, Cytokine Regulating Agents HP228 and HP466, ICAM-1 antisense phosphorothioate oligodeoxynucleotides, soluble complement receptor 1, prednisone, orgotein, glycosaminoglycan polysulphate, minocycline, anti-IL2R antibodies, marine lipids, botanical lipids, auranofin, phenylbutazone, meclofenamic acid, flufenamic acid, zileuton, mycophenolic acid, tacrolimus, sirolimus, amiprilose, cladribine, azaribine, budenoside, epidermal growth factor, aminosalicylates, 6-mercaptopurine, metronidazole, lipoxygenase inhibitors, mesalamine, olsalazine, balsalazide, antioxidants, thromboxane inhibitors, IL-1 receptor antagonists, anti-IL-1β monoclonal antibodies, anti-IL-6 monoclonal antibodies, growth factors, elastase inhibitors, pyridinyl-imidazole compounds, glucuronide-conjugated prodrugs of prednisolone, dexamethasone or budesonide, dextran-conjugated prodrugs of prednisolone, dexamethasone or budesonide, soluble complement receptor 1, slow-release mesalazine, antagonists of Platelet Activating Factor (PAF), ciprofloxacin, lignocaine, prednisolone, methylprednisolone, cyclophosphamide, 4-aminopyridine, tizanidine, interferon-β1a, interferon-β1b, Copolymer 1, hyperbaric oxygen, intravenous immunoglobulin, clabribine, hypertonic saline solutions, antibiotics, continuous hemofiltration, carbapenems, antagonists of TNFα, antagonists of IL-1β, antagonists of IL-6 antagonists of IL-8, SK&F 107647, tetravalent guanylhydrazone CNI-1493, Tissue Factor Pathway Inhibitor, PHP, iron chelators and chelates, diethylenetriamine pentaacetic acid-iron (III) complex, lisofylline, PGG-Glucan, apolipoprotein A-1 reconstituted with lipids, chiral

hydroxamic acids, anti-endotoxin antibodies, E5531, $rBPI_{21}$, Synthetic Anti-Endotoxin Peptides, surfactant replacement therapy and anti-IL-8 antibodies.

123. **(New)** The method of claim 84, wherein the additional therapeutic agent is selected from the group consisting of methotrexate, an IL-12 antibody, leflunomide, Naproxen, Diclofenac, Sulfasalazine, chloroquine, hydroxychloroquine, non-steroidal anti-inflammatory drugs, prednisone, prednisolone, methylprednisolone, and gold.

124. **(New)** The method of claim 84, wherein the additional therapeutic agent is selected from the group consisting of thalidomide, tranexamic acid, T-614, prostaglandin E1, tenidap, meloxicam, piroxicam, indomethacin, azathioprine, ICE inhibitors, zap-70 inhibitors, lck inhibitors, VEGF inhibitors, VEGF-R inhibitors, corticosteroids, and TNF-convertase inhibitors.

125. **(New)** The method of claim 84, wherein the additional therapeutic agent is selected from the group consisting of interleukin-11, interleukin-13, interleukin-17 inhibitors, penicillamine, chloroquine, hydroxychloroquine, chlorambucil, cyclophosphamide, cyclosporin, anti-thymocyte globulin, anti-CD4 antibodies, CD5-toxins, orally-administered collagen, lobenzarit disodium, cytokine regulating agents HP228 and HP 466, ICAM-1 antisense phosphorothioate oligodeoxynucleotides, soluble complement receptor 1, orgotein, glycosaminoglycan polysulphate, minocycline, anti-IL2R antibodies, marine lipids, botanical lipids, auranofin, phenylbutazone, meclofenamic acid, and flufenamic acid.

126. **(New)** The method of claim 84, wherein the additional therapeutic agent is selected from the group consisting of zileuton, mycophenolic acid, tacrolimus, sirolimus, amiprilose, cladribine, azaribine, budenoside, epidermal growth factor, aminosalicylates, 6-mercaptopurine, metronidazole, lipoxygenase inhibitors, mesalamine, olsalazine, balsalazide, antioxidants, thromboxane inhibitors, IL-1 receptor antagonists, anti-IL-1β monoclonal antibodies, anti-IL-6 monoclonal antibodies, growth factors, elastase inhibitors, pyridinyl-imidazole compounds, glucuronide-conjugated prodrugs of prednisolone, dexamethasone or budesonide, dextran-conjugated prodrugs of prednisolone, dexamethasone or budesonide,

soluble complement receptor 1, slow-release mesalazine, antagonists of Platelet Activating Factor (PAF), ciprofloxacin, and lignocaine.

127. **(New)** The method of claim 84, wherein the additional therapeutic agent is selected from the group consisting of cyclophosphamide, 4-aminopyridine, tizanidine, interferon-β1a, interferon-β1b, Copolymer 1, hyperbaric oxygen, intravenous immunoglobulin, clabribine, hypertonic saline solutions, antibiotics, continuous hemofiltration, carbapenems, antagonists of TNFα, antagonists of IL-1β, antagonists of IL-6 antagonists of IL-8, SK&F 107647, tetravalent guanylhydrazone CNI-1493, Tissue Factor Pathway Inhibitor, PHP, iron chelators and chelates, diethylenetriamine pentaacetic acid-iron (III) complex, lisofylline, PGG-Glucan, apolipoprotein A-1 reconstituted with lipids, chiral hydroxamic acids, anti-endotoxin antibodies, E5531, $rBPI_{21}$, Synthetic Anti-Endotoxin Peptides, surfactant replacement therapy and anti-IL-8 antibodies.

128. **(New)** The method of claim 89, wherein the additional therapeutic agent is selected from the group consisting of methotrexate, an IL-12 antibody, leflunomide, Naproxen, Diclofenac, Sulfasalazine, chloroquine, hydroxychloroquine, non-steroidal anti-inflammatory drugs, prednisone, prednisolone, methylprednisolone, and gold.

129. **(New)** The method of claim 99, wherein the additional therapeutic agent is selected from the group consisting of methotrexate, an IL-12 antibody, leflunomide, Naproxen, Diclofenac, Sulfasalazine, chloroquine, hydroxychloroquine, non-steroidal anti-inflammatory drugs, prednisone, prednisolone, methylprednisolone, and gold.

130. **(New)** The method of claim 99, wherein the additional therapeutic agent is selected from the group consisting of thalidomide, tranexamic acid, T-614, prostaglandin E1, tenidap, meloxicam, piroxicam, indomethacin, azathioprine, ICE inhibitors, zap-70 inhibitors, lck inhibitors, VEGF inhibitors, VEGF-R inhibitors, corticosteroids, and TNF-convertase inhibitors.

131. **(New)** The method of claim 99, wherein the additional therapeutic agent is selected from the group consisting of interleukin-11, interleukin-13, interleukin-17 inhibitors, penicillamine, chloroquine, hydroxychloroquine, chlorambucil, cyclophosphamide, cyclosporin, anti-thymocyte globulin, anti-CD4 antibodies, CD5-toxins, orally-administered collagen, lobenzarit disodium, cytokine regulating agents HP228 and HP 466, ICAM-1 antisense phosphorothioate oligodeoxynucleotides, soluble complement receptor 1, orgotein, glycosaminoglycan polysulphate, minocycline, anti-IL2R antibodies, marine lipids, botanical lipids, auranofin, phenylbutazone, meclofenamic acid, and flufenamic acid.

132. **(New)** The method of claim 99, wherein the additional therapeutic agent is selected from the group consisting of zileuton, mycophenolic acid, tacrolimus, sirolimus, amiprilose, cladribine, azaribine, budenoside, epidermal growth factor, aminosalicylates, 6-mercaptopurine, metronidazole, lipoxygenase inhibitors, mesalamine, olsalazine, balsalazide, antioxidants, thromboxane inhibitors, IL-1 receptor antagonists, anti-IL-1β monoclonal antibodies, anti-IL-6 monoclonal antibodies, growth factors, elastase inhibitors, pyridinyl-imidazole compounds, glucuronide-conjugated prodrugs of prednisolone, dexamethasone or budesonide, dextran-conjugated prodrugs of prednisolone, dexamethasone or budesonide, soluble complement receptor 1, slow-release mesalazine, antagonists of Platelet Activating Factor (PAF), ciprofloxacin, and lignocaine.

133. **(New)** The method of claim 99, wherein the additional therapeutic agent is selected from the group consisting of cyclophosphamide, 4-aminopyridine, tizanidine, interferon-β1a, interferon-β1b, Copolymer 1, hyperbaric oxygen, intravenous immunoglobulin, clabribine, hypertonic saline solutions, antibiotics, continuous hemofiltration, carbapenems, antagonists of TNFα, antagonists of IL-1β, antagonists of IL-6 antagonists of IL-8, SK&F 107647, tetravalent guanylhydrazone CNI-1493, Tissue Factor Pathway Inhibitor, PHP, iron chelators and chelates, diethylenetriamine pentaacetic acid-iron (III) complex, lisofylline, PGG-Glucan, apolipoprotein A-1 reconstituted with lipids, chiral hydroxamic acids, anti-endotoxin antibodies, E5531, $rBPI_{21}$, Synthetic Anti-Endotoxin Peptides, surfactant replacement therapy and anti-IL-8 antibodies.

134. **(New)** The method of claim 104, wherein the additional therapeutic agent is selected from the group consisting of methotrexate, an IL-12 antibody, leflunomide, Naproxen, Diclofenac, Sulfasalazine, chloroquine, hydroxychloroquine, non-steroidal anti-inflammatory drugs, prednisone, prednisolone, methylprednisolone, and gold.

135. **(New)** A method for treating a subject suffering from rheumatoid arthritis, comprising administering to the subject an antibody and at least one additional therapeutic agent, such that the rheumatoid arthritis is treated, wherein the antibody is an isolated human antibody, or an antigen-binding portion thereof, that dissociates from human TNFα with a K_d of 1×10^{-8} M or less and a K_{off} rate constant of 1×10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1×10^{-7} M or less.

136. **(New)** The method of claim 135, wherein the additional therapeutic agent is selected from the group consisting of methotrexate, an IL-12 antibody, leflunomide, Naproxen, Diclofenac, Sulfasalazine, chloroquine, hydroxychloroquine, non-steroidal anti-inflammatory drugs, prednisone, prednisolone, methylprednisolone, and gold.

137. **(New)** The method of claim 135, wherein the wherein the additional therapeutic agent is selected from the group consisting of cytokine suppressive anti-inflammatory drugs, CDP-571/BAY-10-3356, cA2, 75 kdTNFR-IgG , 55 kdTNFR-IgG, IDEC-CE9.1/SB 210396, DAB 486-IL-2, DAB 389-IL-2, Anti-Tac, IL-4, IL-10, IL-4 agonists, IL-10 agonists, IL-1RA, TNF-bp/s-TNFR, S284, R973401, MK-966, Iloprost, thalidomide, tranexamic acid, T614, prostaglandin E1, Tenidap, Naproxen, Meloxicam, Piroxicam, Indomethacin, Azathioprine, ICE inhibitors, zap-70 inhibitors, lck inhibitors, VEGF inhibitors, VEGF-R inhibitors, corticosteroids, TNF-convertase inhibitors, anti-IL-12 antibodies, interleukin-11, interleukin-13, interleukin-17 inhibitors, penicillamine, chlorambucil, cyclophosphamide, cyclosporin, anti-thymocyte globulin, anti-CD4 antibodies, CD5-toxins, orally-administered collagen, lobenzarit disodium, Cytokine Regulating Agents HP228 and HP466, ICAM-1 antisense phosphorothioate oligodeoxynucleotides, soluble complement receptor 1, orgotein, glycosaminoglycan polysulphate, minocycline, anti-IL2R antibodies, marine lipids, botanical

lipids, auranofin, phenylbutazone, meclofenamic acid, flufenamic acid, intravenous immunoglobulin, zileuton, mycophenolic acid, tacrolimus, sirolimus, amiprilose, cladribine, and azaribine.

138. **(New)** A method for treating a subject suffering from rheumatoid arthritis, comprising administering to the subject an antibody and at least one additional therapeutic agent such that the rheumatoid arthritis is treated, wherein the antibody is an isolated human antibody, or antigen-binding portion thereof, with the following characteristics:

a) dissociates from human TNFα with a K_{off} rate constant of 1×10^{-3} s^{-1} or less, as determined by surface plasmon resonance;

b) has a light chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 3, or modified from SEQ ID NO: 3 by a single alanine substitution at position 1, 4, 5, 7 or 8 or by one to five conservative amino acid substitutions at positions 1, 3, 4, 6, 7, 8 and/or 9;

c) has a heavy chain CDR3 domain comprising the amino acid sequence of SEQ ID NO: 4, or modified from SEQ ID NO: 4 by a single alanine substitution at position 2, 3, 4, 5, 6, 8, 9, 10 or 11 or by one to five conservative amino acid substitutions at positions 2, 3, 4, 5, 6, 8, 9, 10, 11 and/or 12.

139. **(New)** A method for treating a subject suffering from rheumatoid arthritis, comprising administering to the subject an antibody and at least one additional therapeutic agent, such that the rheumatoid arthritis is treated, wherein the antibody is an isolated human antibody, or an antigen binding portion thereof, with a light chain variable region (LCVR) comprising the amino acid sequence of SEQ ID NO: 1 and a heavy chain variable region (HCVR) comprising the amino acid sequence of SEQ ID NO: 2

140. **(New)** A method for treating a subject suffering from rheumatoid arthritis, comprising administering to the subject an antibody and at least one additional therapeutic agent such that the rheumatoid arthritis is treated, wherein the antibody is D2E7.

141. **(New)** A method for treating a subject suffering from rheumatoid arthritis, comprising administering to the subject an antibody and methotrexate, such that the rheumatoid

arthritis is treated, wherein the antibody is an isolated human antibody, or an antigen-binding portion thereof, that dissociates from human TNFα with a K_d of 1 x 10^{-8} M or less and a K_{off} rate constant of 1 x 10^{-3} s^{-1} or less, both determined by surface plasmon resonance, and neutralizes human TNFα cytotoxicity in a standard *in vitro* L929 assay with an IC_{50} of 1 x 10^{-7} M or less.

142. **(New)** A method for treating a subject suffering from rheumatoid arthritis, comprising administering to the subject an antibody and methotrexate such that the rheumatoid arthritis is treated, wherein the antibody is D2E7.